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A Giant Intra-Abdominal Plasmablastic Neoplasm Favoring Lymphoma Mimicking an Exacerbation of Severe Fistulizing Crohn's Disease: A Case Report

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Patient: Female, 44-year-old
Final Diagnosis: Plasmablastic lymphoma
Symptoms: Ascites • cahexia • CD exacerbation • tumor
Clinical Procedure: Immunotherapy
Specialty: Gastroenterology and Hepatology
Objective: Rare coexistence of disease or pathology

Background: Plasmablastic lymphoma (PBL) is a rare, aggressive large B-cell lymphoma associated with immunosuppression. In Crohn's disease, it can mimic disease exacerbation or infection. We describe a rapidly enlarging intra-abdominal plasmablastic neoplasm in a medically complex patient, emphasizing diagnostic vigilance without inferring a causal association with biological therapy.

Case Report: A 44-year-old woman with severe fistulizing Crohn's disease treated sequentially with adalimumab, infliximab, and ustekinumab developed progressive systemic deterioration despite partial intestinal stabilization. Her course included portal hypertension secondary to portal vein thrombosis, refractory ascites, severe malnutrition, and recurrent systemic and multidrug-resistant infections. In September 2023 imaging showed a rapidly enlarging intra-abdominal/pelvic mass measuring more than 22 cm. Core-needle biopsy demonstrated a plasmablastic neoplasm positive for CD38, CD138, MUM1, and c-MYC, with equivocal CD10 and negative CD45, favoring PBL. Epstein–Barr virus-encoded RNA in situ hybridization, CD20, PAX5, Ki-67, anaplastic lymphoma kinase, human herpesvirus 8, and tissue light-chain restriction were unavailable. Bone marrow examination, serum and urine immunofixation, complete monoclonal-protein assessment, and whole-body skeletal imaging were not performed; therefore, plasmablastic myeloma could not be definitively excluded. Intensive chemotherapy was not feasible because her Eastern Cooperative Oncology Group performance status was at least 3 and she had multiorgan dysfunction, severe malnutrition, and active infections. The patient was transitioned to palliative care and subsequently died.

Conclusions: Discordance between apparent intestinal control and systemic deterioration should prompt urgent tissue diagnosis. The findings favor PBL within the spectrum of plasmablastic neoplasms but neither establish drug-specific causation nor definitively exclude plasmablastic myeloma.

Keywords: Crohn Disease • Plasmablastic Lymphoma • Immunosuppression Therapy • Ustekinumab • Ascites • Gastroenterology • Lymphoma, Large B-Cell, Diffuse • Immunosuppression • Case Reports
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Introduction

Crohn's disease (CD) is a chronic inflammatory bowel disease characterized by segmental, transmural inflammation that can involve any part of the gastrointestinal tract [1]. Severe and fistulizing disease may require prolonged immunomodulatory and biological therapy, and the therapeutic goal is to achieve and maintain clinical, endoscopic, and histologic remission [2,3]. Mesenchymal stem/stromal cell-based therapy has also been investigated for complex perianal fistulizing Crohn's disease, including local administration of allogeneic umbilical cord-derived cells [4].

Plasmablastic lymphoma (PBL) is a rare, highly aggressive large B-cell lymphoma with terminal plasmacytic differentiation, most commonly occurring in the setting of impaired immune function [5,6]. Its typical immunophenotype includes absent or weak CD20 and PAX5 expression, positivity for plasma cell markers such as CD38, CD138, and MUM1, and a high proliferative index [6,7].

The published literature on PBL in inflammatory bowel disease remains limited to isolated case reports and small series [8-14]. In Crohn's disease, reported sites have included paravertebral soft tissue and skin [8], the colon and small bowel [9,10], and lesions arising in or adjacent to chronic perianal or enterocutaneous fistulas [11,13,14]; PBL has also been described during immunosuppressive treatment of ulcerative colitis [12]. Several recent reports have particularly emphasized fistula-associated lesions that were initially interpreted as chronic inflammatory disease [11,13,14].

In contrast, the present patient developed a giant, rapidly enlarging solid-cystic intraperitoneal/pelvic mass in the setting of portal hypertension caused by portal vein thrombosis, massive refractory ascites, severe malnutrition, and recurrent systemic and multidrug-resistant infections. These overlapping conditions initially favored inflammatory or infectious explanations and obscured recognition of the malignancy despite partial stabilization of intestinal disease. The purpose of this report is therefore diagnostic rather than etiologic: to illustrate when discordance between intestinal activity and systemic deterioration should trigger urgent tissue diagnosis.

Case Report

The patient was a 44-year-old woman with severe fistulizing Crohn's disease diagnosed in 2005 and treated at our tertiary center since December 2020. Ethnicity is not reported because it was not clinically relevant and is omitted to protect patient privacy. No relevant history of tobacco use, alcohol consumption, or substance abuse was documented.

This case report aims to describe the diagnostic challenge of distinguishing progressive inflammatory or infectious complications of Crohn's disease from an aggressive lymphoproliferative malignancy in a chronically immunosuppressed and medically fragile patient. The main educational problem was recognition of a plasmablastic neoplasm favoring PBL presenting as a rapidly enlarging intra-abdominal/pelvic lesion in the setting of severe CD, refractory ascites, cachexia, and recurrent infections.

For clarity, the key chronology was as follows: Crohn's disease was diagnosed in 2005; the patient was referred to our tertiary center in December 2020; adalimumab induction was given from October to November 2020 without remission; infliximab was used from March to June 2021 and interrupted because of suspected tuberculosis, which was not confirmed; infliximab re-exposure in late 2021 caused anaphylaxis; ustekinumab was introduced in early 2022; the last documented maintenance dose was administered in June 2023; further dosing was withheld during the August–September 2023 hospitalization because of pleural empyema and an intra-abdominal collection/mass; -needle biopsy identified a plasmablastic neoplasm favoring PBL in September 2023; and palliative management was implemented after multidisciplinary assessment in November 2023.

Crohn's disease treatment and related procedures

Induction therapy with adalimumab (generic name: adalimumab; subcutaneous administration; administered from October to November 2020 according to the applicable treatment program schedule) failed to induce remission. From March to June 2021, infliximab (generic name: infliximab; intravenous administration) was administered but discontinued after radiological suspicion of tuberculosis; active infection was not confirmed. Re-exposure to infliximab in late 2021 resulted in a severe anaphylactic reaction after the second dose, permanently precluding further therapy with tumor necrosis factor-α (TNF-α) antagonists.

In early 2022, ustekinumab (generic name: ustekinumab; intravenous induction followed by subcutaneous maintenance according to the national drug program schedule) was initiated. Disease activity remained moderate, with Crohn's Disease Activity Index (CDAI) values of approximately 200 to 341. Ustekinumab was clinically well tolerated and led to partial stabilization of intestinal disease activity; however, the patient's systemic condition continued to deteriorate, creating a discordance between apparent intestinal control and the overall clinical trajectory. The last documented maintenance dose was administered in June 2023, and subsequent dosing was withheld during the August–September 2023 hospitalization because of infectious contraindications, including pleural empyema and an intra-abdominal collection.

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Co-Existing Diseases and Complications

The clinical course was complicated by severe protein-calorie malnutrition severe protein-calorie malnutrition progressing to cachexia (body mass index approximately 17 kg/m^2, within the underweight range), hypoalbuminemia, and refractory ascites requiring total parenteral nutrition, oral nutritional support, albumin supplementation, and repeated large-volume paracenteses. Imaging showed entero-enteric fistulae involving the ileocecal, ileotransverse, and ileosigmoid regions.

Chronic iron-deficiency anemia with hemoglobin levels of approximately 7 to 9 g/dL required repeated packed red blood cell transfusions and intravenous iron supplementation. Additional clinically relevant comorbidities and complications included portal vein thrombosis with secondary portal hypertension, grade II esophageal varices, splenomegaly, progressive ascites, and right iliofemoral deep vein thrombosis.

The patient also experienced recurrent serious infections and colonization with multidrug-resistant organisms, including recurrent urinary tract infections, recurrent *Clostridioides difficile* infection requiring fecal microbiota transplantation, catheter-related bloodstream infections with septic shock, polymicrobial sepsis, infective endocarditis with right atrial vegetation, disseminated intravascular coagulation, and acute cardiorespiratory failure. These complications contributed to cumulative systemic vulnerability. A summary of infections is provided in **Table 1**, and therapeutic interventions are summarized in **Table 2**.

Diagnostic Evolution, Final Diagnosis, and Clinical Solution

In 2023, intestinal disease activity appeared partially stabilized, but systemic cachexia and refractory ascites progressed after prior ustekinumab exposure and during a period in which further ustekinumab dosing was withheld because of infectious contraindications. In September 2023, cross-sectional imaging revealed a large intraperitoneal/pelvic mass. A newly identified, well-demarcated peritoneal fluid collection on computed tomography was subsequently visualized on ultrasonography as a septated lesion with multiple internal partitions, raising concern for a proliferative process rather than a simple inflammatory or infectious collection.

Pathological Findings and Differential Diagnosis

CT-guided core-needle biopsy was performed. Available histopathological data showed an infiltrate of polymorphic cells with abundant cytoplasm. Immunohistochemistry demonstrated positivity for CD38, CD138, MUM1, and c-MYC; CD10 expression was equivocal. CD45, cytokeratins (CK/CK7/CK20), CDX2, calretinin, WT-1, TTF-1, ER, p53, inhibin, PLAP, bcl-2, and bcl-6 were negative. The available morphology and immunophenotype favored plasmablastic lymphoma, but EBER, CD20, PAX5, Ki-67, ALK, HHV8, and tissue light-chain restriction were unavailable, and a complete plasma-cell myeloma evaluation could not be performed. Because of an ECOG performance status of at least 3, multiorgan dysfunction, severe malnutrition, and active infectious complications, the patient was not eligible for intensive chemotherapy, was transitioned to palliative care, and subsequently died. The available biopsy established a plasmablastic neoplasm and, in the clinical context, favored plasmablastic lymphoma. It did not, however, permit a definitive distinction from plasmablastic myeloma. On 2 November 2023, serum free kappa and lambda light chains were elevated at 83.98 mg/L and 63.50 mg/L, respectively; the calculated kappa/lambda ratio was 1.32. Total calcium was 8.76 mg/dL. A non-contrast head CT performed on the same day showed a solitary, nonspecific 12 × 7 mm osteolytic focus in the left frontal bone. Bone marrow examination, serum and urine immunofixation, contemporaneous complete monoclonal-protein assessment, and dedicated whole-body skeletal imaging were not performed. Accordingly, these available findings neither confirmed nor excluded plasma-cell myeloma. The pathology documentation accessible to the authors did not contain results for EBER-ISH, CD20, PAX5, Ki-67, ALK, HHV8, or tumor kappa/lambda light-chain restriction. Consequently, EBV association, the complete B-cell marker profile, proliferative index, tissue clonality, and exclusion of selected alternative plasmablastic entities could not be established. Ascitic fluid parameters and tumor markers were nonspecific during the observation period and are summarized in **Table 3**.

Plasmablastic myeloma and extramedullary plasmacytoma were considered in the differential diagnosis. However, because of the patient's poor performance status, multiorgan dysfunction, infectious burden, and rapidly deteriorating condition, a complete plasma-cell myeloma workup was not feasible. Bone marrow examination, serum and urine immunofixation with complete monoclonal-protein assessment, and dedicated whole-body skeletal imaging for osteolytic lesions were not completed. Available late serum free light-chain abnormalities alone were insufficient to definitively exclude or confirm plasma-cell myeloma. Therefore, the diagnosis should be interpreted in the context of the available core-needle biopsy morphology, immunophenotype, clinical presentation, and acknowledged diagnostic limitations.

Because of extremely poor performance status (Eastern Cooperative Oncology Group [ECOG] ≥ 3), multiorgan dysfunction, severe malnutrition, and a high anticipated burden of cytotoxic treatment, aggressive chemotherapy was considered inappropriate after multidisciplinary hematology assessment. Supportive and palliative care were implemented. In November 2023, the patient developed severe SARS-CoV-2

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Table 1. Infections during the observation and treatment period.

Period	Specimen/site	Pathogen	Resistance profile	Clinical significance	Treatment
Jan 2021	Respiratory tract	SARS-CoV-2	–	Previous SARS-CoV-2 infection	Symptomatic treatment
Apr 2021	Urine	*Klebsiella pneumoniae*	ESBL-negative, susceptible	UTI	Oral fosfomycin (according to susceptibility testing)
Jun 2021	Blood (catheter)	*Klebsiella pneumoniae*	–	Catheter-related bloodstream infection	Antibiotic therapy and catheter removal
Aug–Nov 2021	Blood	Actinomyces odontolyticus, Rothia dentocariosa	–	Polymicrobial sepsis	Intravenous antibiotic therapy
Aug–Nov 2021	Ascitic fluid	*Escherichia coli*	ESBL-negative, susceptible	Spontaneous bacterial peritonitis (SBP)	Targeted antibiotic therapy
Aug–Nov 2021	Urine	*E. coli, Proteus mirabilis*	ESBL-negative	Recurrent UTI	Antibiotic therapy according to susceptibility testing
Sep 2021	Gastrointestinal tract	*Clostridioides difficile*	Toxigenic strain	Recurrent CDI	Oral vancomycin; fecal microbiota transplantation (FMT)
Sep 2021	Perianal region	Mixed bacterial flora	–	Post-antibiotic dysbiosis	Conservative management
Nov 2021	Urine	*Proteus mirabilis*	–	UTI	Ciprofloxacin with metronidazole
Dec 2021	Perianal region (screening)	*Klebsiella pneumoniae*	KPC+/MBL+	Colonization with multidrug-resistant organism (MDRO)	Epidemiological isolation
Feb 2023	Urine	*Escherichia coli*	ESBL+, carbapenem-susceptible	UTI – multidrug-resistant organism (MDRO)	Carbapenem or amikacin (according to susceptibility testing)
Sep 2023	Pleural and peritoneal fluid	No growth (culture-negative)	–	Pleural empyema, intra-abdominal fluid collection	Surgical drainage
Sep 2023	Postoperative wound	*Acinetobacter baumannii*	MDR, carbapenem-resistant	Surgical site infection	Targeted antibiotic therapy
Sep 2023	Postoperative wound	*Enterococcus faecalis* + *Proteus mirabilis*	HLAR (high-level aminoglycoside resistance) / ESBL - negative	Mixed-etiology wound infection	Targeted antibiotic therapy
Nov 2023	Urine	Escherichia coli	ESBL+, fluoroquinolone-resistant	Recurrent UTI	Antibiotic therapy according to susceptibility testing

Abbreviations: "–" indicates missing or unavailable data; ESBL, extended-spectrum beta-lactamase; KPC, Klebsiella pneumoniae carbapenemase; MBL, metallo-beta-lactamase; MDR, multidrug-resistant; HLAR, high-level aminoglycoside resistance; UTI, urinary tract infection; FMT, fecal microbiota transplantation; MDRO, multidrug-resistant organism.

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Table 2. Therapeutic interventions according to the underlying clinical problem.

Type of intervention	Clinical indications	Medications / measures	Clinical effectiveness	Safety considerations
Treatment of IBD exacerbations and maintenance therapy	Crohn's disease flare	Systemic corticosteroids (hydrocortisone, prednisone); adalimumab; infliximab; ustekinumab	Adalimumab ineffective; infliximab discontinued due to anaphylaxis; ustekinumab achieved disease stabilization	Anaphylactic reaction to infliximab; ustekinumab well tolerated
Antibiotic therapy for infections (sepsis, UTI)	Catheter-related sepsis; urinary tract infections	Meropenem plus linezolid; ciprofloxacin plus metronidazole; fosfomycin	Clinical improvement with reduction in CRP and procalcitonin levels	Risk of dysbiosis and recurrent CDI; Close monitoring for adverse effects is required
Treatment of CDI and intestinal dysbiosis	Recurrent *Clostridioides difficile* infection	Oral vancomycin; fecal microbiota transplantation (FMT)	Symptomatic improvement after FMT; reduction in recurrence	Risk of CDI recurrence; need to minimize antibiotic exposure
Prevention and control of multidrug-resistant organisms (MDROs)	Colonization with KPC/MBL-producing Klebsiella pneumoniae	Epidemiological isolation; antibiotic stewardship	Reduced transmission of multidrug-resistant organisms	Strict antimicrobial stewardship required
Hemodynamic support and management of catheter-related sepsis	Septic shock; catheter-related bloodstream infection	Norepinephrine; therapeutic paracenteses; central venous catheter replacement	Hemodynamic stabilization	Risk of catheter-related infections; strict aseptic technique and fluid balance monitoring required
Parenteral nutrition (PN) and oral nutritional support (ONS)	Severe cachexia; hypoalbuminemia; refractory ascites	Commercial parenteral nutrition formulations (Olimel, SmofKabiven, etc.); amino acid solutions; vitamin and trace element supplementation; albumin	Improved nutritional status with increased serum albumin and body weight	Risk of metabolic complications and catheter-related infections
Treatment of anemia	Iron-deficiency anemia (Hb 7-9 g/dL)	Packed red blood cell transfusions (≥ 6 units); intravenous iron (ferric derisomaltose)	Improvement in hemoglobin levels and iron stores	Risk of transfusion reactions and iron overload
Management of ascites and portal hypertension complications	Portal hypertension; ascites; prevention of variceal bleeding	Furosemide; spironolactone; chlorthalidone; propranolol; therapeutic paracenteses	Reduction of edema and improved ascites control	Risk of electrolyte imbalance and hyponatremia
Supportive pharmacotherapy	Blood pressure control; edema; esophageal varices; circulatory support	Diuretics; norepinephrine; beta-blockers; proton pump inhibitors; probiotics	Hemodynamic stabilization	Close monitoring of electrolytes and adverse drug reactions is required

Abbreviations: IBD, inflammatory bowel disease; UTI, urinary tract infection; CDI, *Clostridioides difficile* infection; CRP, C-reactive protein; PN, parenteral nutrition; ONS, oral nutritional support.

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Table 3. Laboratory parameters of ascitic fluid.

Date of paracentesis	Serum albumin (g/dL)	Ascitic fluid albumin (g/dL)	SAAG (g/dL)	Ascitic fluid total protein (g/dL)	Ascitic fluid LDH (U/L)	Ascitic fluid glucose (mg/dL)	Ascitic fluid amylase (U/L)	PMN count (cells/μL)	Remarks
2020-12-23	2.82	0.7	2.12	0.70	47.4	79.4	38.9	33	Minimal sediment
2021-01-08	2.21	0.7	1.51	0.32	29.1	121.0	51.7	–	Numerous red blood cells
2021-01-14	2.04	–	–	0.23	24.4	116.0	80.1	–	Few bacteria observed on microscopy
2021-01-19	–	–	–	–	–	–	–	–	CA-125 769 U/mL
2021-04-24	2.54	0.024	2.51	0.25	27.1	121.0	21.2	36	–
2021-09-10	–	–	–	–	36.6	118.0	16.0	–	–
2021-09-22	–	–	–	–	49.4	136.0	21.5	–	–
2021-10-19	2.61	0.7	1.91	–	64.8	101.0	21.2	8	CA-125 1121 U/mL
2021-10-26	–	–	–	–	127.0	123.0	30.6	162	–
2022-01-10	2.89	0.6	2.29	1.19	127.0	51.6	19.1	81	CEA 1.63 ng/mL; CA 19-9 2.62 U/mL
2023-04-13	2.69	1.1	1.59	2.34	99.2	136.0	22.5	–	CEA 0.84 ng/mL; CA 19-9 2.29 U/mL
2023-09-11	3.43	1.4	2.03	3.39	51.3	–	23.5	68	Negative microbiological cultures; CEA 1.49 ng/mL; CA 19-9 3.81 U/mL; CA-125 231.0 U/mL

Abbreviations: SAAG, serum–ascites albumin gradient; PMN, polymorphonuclear leukocyte count; LDH, lactate dehydrogenase; CEA, carcinoembryonic antigen; CA 19-9, carbohydrate antigen 19-9; CA-125, cancer antigen 125. "–" indicates missing or unavailable data.

infection with bilateral pneumonia. Following stabilization, the hematology board concluded that no curative oncologic options were feasible, and the patient was referred to long-term palliative care. She subsequently died after transition to palliative management.

Discussion

This case illustrates the central diagnostic pitfall of the report: in severe Crohn's disease, an aggressive lymphoma may be masked by chronic inflammation, ascites, abscess-like collections, malnutrition, and recurrent infection. PBL is rare but highly aggressive, and its presentation can overlap with inflammatory bowel disease exacerbation or infectious complications [6,15].

In this patient, a plasmablastic neoplasm favoring PBL was identified after sequential biologic exposure, including prior anti-TNF therapy and ustekinumab; however, temporal coexistence should not be interpreted as causation. This single case cannot attribute PBL to ustekinumab. In patients with inflammatory bowel disease, thiopurines and TNF-α antagonists have been associated with a moderate but clinically significant increase in lymphoma risk, with the highest risk reported during combination therapy [16,17]. Ustekinumab has not been conclusively associated with increased lymphoma risk in inflammatory bowel disease, and no clear signal for increased lymphoma or overall malignancy risk has emerged from long-term pooled phase 2/3 clinical-trial safety data [18]. Therefore, the present observation should be viewed as a diagnostic-vigilance signal in a vulnerable host rather than as evidence of a treatment-specific lymphoma risk.

The key clinical implication is diagnostic rather than etiologic: clinical improvement or stabilization under biologic therapy should not reduce oncologic vigilance. A rapidly enlarging

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Table 4. Differential diagnosis between plasmablastic lymphoma and plasmablastic myeloma in the present case.

Case-specific interpretation based exclusively on the records available to the authors.

Diagnostic domain	Available findings in this patient	Differential interpretation
Morphology	Infiltrate of plasmablastic/polymorphic cells with abundant cytoplasm	Consistent with a plasmablastic neoplasm, but morphology alone does not distinguish PBL from PBM
Plasma-cell phenotype	CD38+, CD138+, MUM1+, and c-MYC+; CD10 equivocal; CD45-	Confirms plasmacytic differentiation but is not specific for PBL; a similar phenotype may occur in PBM
B-cell markers	CD20 and PAX5 results were not available in the documentation accessible to the authors	The B-cell marker profile cannot be assessed. Missing results neither confirm nor exclude PBL
EBV association	EBER-ISH was unavailable. Historical EBV serology does not establish EBV infection in tumor cells	Tumor EBV status cannot be determined. EBER positivity would support PBL, but its unavailability is not discriminatory
Proliferation and exclusion markers	Ki-67, ALK, and HHV8 results were unavailable	The expected high proliferative index cannot be documented, and selected alternative plasmablastic entities cannot be excluded
Clonality and serum free light chains	Tumor kappa/lambda restriction was unavailable. On 2 November 2023, serum kappa was 83.98 mg/L and lambda 63.50 mg/L; the calculated kappa/lambda ratio was 1.32. The assay-specific reference range was unavailable	Tissue clonality is unproven. Concurrent elevation of both serum light chains, without an interpretable assay-specific ratio, does not establish a definitive monoclonal pattern
Bone findings	Head CT on 2 November 2023 showed a solitary, nonspecific 12 × 7 mm osteolytic focus in the left frontal bone; dedicated whole-body skeletal imaging was not performed	The finding increases concern for PBM but is neither specific nor sufficient to establish myeloma-related bone disease
Myeloma workup	Bone marrow examination, serum and urine immunofixation, contemporaneous complete monoclonal-protein assessment, and dedicated whole-body skeletal imaging were not performed	PBM cannot be definitively excluded because the required clinicopathological assessment is incomplete
Clinical pattern	Giant extraosseous intra-abdominal/pelvic mass; total calcium 8.76 mg/dL; no documented hypercalcemia. Severe inflammation, infection, and malnutrition provide alternative explanations for anemia and systemic deterioration	The bulky extraosseous presentation may favor PBL, but extramedullary PBM remains possible; no clinical feature is independently decisive
Overall interpretation	The available morphology and immunophenotype establish a plasmablastic neoplasm	The findings favor PBL in the clinical context, but the incomplete tissue panel and myeloma workup preclude definitive distinction from PBM

Abbreviations: PBL, plasmablastic lymphoma; PBM, plasmablastic myeloma; EBER-ISH, Epstein–Barr virus-encoded RNA in situ hybridization; EBV, Epstein–Barr virus; CT, computed tomography.

mass, atypical fluid collection, treatment-refractory lesion, or discordance between clinical deterioration and presumed inflammatory activity should prompt urgent histopathological evaluation. Radiology and inflammatory biomarkers alone are insufficient to reliably distinguish infection, active CD, and malignancy in this setting [19-21].

Inflammatory markers were also limited in this case. C-reactive protein (CRP) may be elevated in active CD, infection, or malignancy, while procalcitonin (PCT) is more specific for systemic bacterial infection. However, normal PCT levels do not exclude malignancy or other noninfectious causes of systemic deterioration [22,23]. Similarly, ascitic fluid abnormalities, including reduced glucose and elevated lactate dehydrogenase, may suggest malignant involvement, but lack sufficient specificity without cytological or histopathological confirmation [24].

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The interpretation of tumor markers in patients with ascites also requires caution. CA-125 reflects serosal irritation and may be elevated in non-gynecologic conditions, including lymphoma with serosal effusion [25-27]. Therefore, a positive Risk of Ovarian Malignancy Algorithm (ROMA) result in women with chronic effusions should be interpreted as a signal for broader oncologic evaluation rather than as evidence of ovarian carcinoma alone.

The diagnosis of PBL requires integration of clinical presentation, histopathology, and immunophenotyping. In the present case, the available core-needle biopsy morphology and immunophenotype supported plasmablastic lymphoma: CD38, CD138, MUM1, and c-MYC were positive; CD10 was equivocal; and CD45, cytokeratins, CDX2, calretinin, WT-1, TTF-1, ER, p53, inhibin, PLAP, bcl-2, and bcl-6 were negative. Differential diagnosis includes extramedullary plasmacytoma, plasmablastic myeloma, primary effusion lymphoma, ALK-positive large B-cell lymphoma, and other EBV-associated large B-cell lymphomas [6,28]. EBER status and several ancillary markers, including CD20, PAX5, Ki-67, ALK, HHV8, and tumor light-chain restriction, were not available in the retrieved report; this limitation should be explicitly acknowledged. In patients with poor performance status, comprehensive staging and extensive ancillary investigations may be impracticable, as occurred in the present case. These limitations prevent definitive exclusion of plasmablastic myeloma and reduce the certainty of the final classification. The pathology diagnosis is therefore reported as a plasmablastic neoplasm favoring PBL. The educational value of this case lies primarily in the diagnostic pitfall: a giant malignant lesion was initially obscured by portal-hypertension-related ascites, chronic inflammation, severe infection, and profound malnutrition. Case-specific interpretation based exclusively on the records available to the authors is presented in **Table 4**.

Treatment decisions in PBL depend strongly on performance status and disease extent. Intensive regimens such as dose-adjusted EPOCH, sometimes combined with novel agents, may improve outcomes in selected patients with preserved functional status [29-31]. In the present patient, severe Crohn's disease complications, profound cachexia, multiorgan dysfunction, and infectious burden made intensive chemotherapy inappropriate. This limitation is central to the case: delayed recognition of lymphoma in a medically fragile patient may eliminate the possibility of curative-intent therapy [29].

The main limitation of this report is that some diagnostic procedures could not be completed because of the patient's critical condition. EBER in situ hybridization was unavailable, and the comprehensive workup for plasma-cell myeloma—including bone marrow examination, complete monoclonal-protein assessment, and dedicated whole-body skeletal imaging—could not be completed. These limitations reduce diagnostic completeness and should temper the strength of the conclusions. Nevertheless, the case remains clinically relevant as a practice-alert report because it supports lowering the threshold for biopsy and hematology consultation in immunosuppressed patients with inflammatory bowel disease and atypical abdominal findings.

The present case underscores 4 practice-alert messages: (1) apparent intestinal stabilization during biologic therapy does not exclude concurrent malignancy and should not be read as evidence of drug-specific causation; (2) lack of a specific laboratory alarm marker does not rule out aggressive lymphoproliferation; (3) rapid tumor progression can occur over a short interval despite previously unremarkable imaging; (4) management should be multidisciplinary and should balance expected benefit against treatment burden and patient condition.

Conclusions

Plasmablastic lymphoma can be clinically obscured by active inflammation, ascites, infection, and malnutrition in patients with severe Crohn's disease. This single case cannot establish that ustekinumab caused or promoted lymphoma; rather, it illustrates that a lymphoproliferative malignancy can present after sequential biologic exposure in a medically fragile patient with cumulative immunologic and systemic vulnerability.

Therefore, the practical message of this case is an evidence-bounded clinical alert: in severe Crohn's disease under prolonged immunomodulatory or biologic therapy, any atypical or rapidly enlarging intra-abdominal lesion, mass, or fluid collection should lower the threshold for core-needle biopsy and early hematology input, particularly when systemic deterioration is discordant with apparent intestinal disease control. Because EBER status and complete plasma-cell myeloma workup were unavailable, this case should be interpreted as a diagnostic-vigilance report based on available biopsy findings, not as proof of drug-specific causation or as a fully staged hematologic case.

Future studies should define practical surveillance thresholds for malignancy in patients with severe inflammatory bowel disease receiving prolonged immunomodulatory or biologic therapy. Until such thresholds are available, biomarkers and ascitic fluid parameters should be considered supportive only. In medically fragile patients, intensive chemotherapy may not be feasible once performance status deteriorates; therefore, early biopsy and hematology consultation remain the key practical steps when imaging findings or clinical deterioration are atypical.

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Patient Consent

Informed consent for publication of this case report and any accompanying images was obtained from the patient prior to her death. All identifying information has been removed to ensure anonymity.

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